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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
BUNNER, BRIDGET E	
ART UNIT	PAPER NUMBER
1647	

DATE MAILED: 12/14/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/762,594	PAPADOPOULOS ET AL.	
	Examiner	Art Unit	
	Bridget E. Bunner	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *the IDS filed on 10/4/04 and the amendment of 9/27/04*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *10-16,41,43,44,46-52,57-64 and 69-82* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☒ Claim(s) *10-16,41 and 48-52* is/are allowed.

6)☒ Claim(s) *43-44, 46-47, 57-64, 69-82* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *10/4/04*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Status of Application, Amendments and/or Claims

The amendment of 27 September 2004 has been entered in full. Claims 41, 43-44 are amended. Claims 77-82 are added.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claims 10-16, 41, 43-44, 46-52, 57-64, and 69-82 are under consideration in the instant application.

Claim Objections

1. Claim 81 is objected to because of the following informalities: Claim 81, line 2 recites "a polypeptide that is impairs". The word "is" should be deleted. Appropriate correction is required.

Claim Rejections - 35 USC § 112, first paragraph

2. Claims 43-44, 46-47, 57-64, 69-82 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. The basis for this rejection is set forth at pg 3-8 of the previous Office Action (23 June 2004).

The claims are directed to an isolated nucleic acid sequence that is at least 90% identical to a nucleotide sequence encoding SEQ ID NO: 7, which isolated nucleic acid encodes a polypeptide that is capable of regulating progesterone synthesis, or the complement thereof. The claims recited an isolated nucleic acid sequence that is at least 90% identical to a nucleotide

sequence encoding SEQ ID NO: 7, which isolated nucleic acid encodes a polypeptide that impairs cholesterol delivery. The claims recite a process of producing a peripheral-type benzodiazepine-associated protein. The claims recite a vector comprising a heterologous promoter linked the PAP7 nucleotide sequence and reagents comprising the nucleic acid. The claims recite an isolated nucleic acid that hybridizes to the complement of SEQ ID NO: 2 or to the complement of the nucleotide sequence that encodes the polypeptide of SEQ ID NO: 7. The claims recited an isolated nucleic acid sequence that is at least 90% identical to a nucleotide sequence encoding SEQ ID NO: 7, which isolated nucleic acid encodes a polypeptide that increases cholesterol delivery.

Applicant's arguments (27 September 2004), as they pertain to the rejections have been fully considered but are not deemed to be persuasive for the following reasons.

(i) Applicant asserts that one or ordinary skill in the art in possession of the specification and knowledge available to the art would be apprised of how to make and identify the claimed nucleic acid sequences. Applicant indicates that the specification discloses how PAPs may be identified and how variants may be made. Applicant cites, for example, Sher et al. and Kopchick et al. Applicant also submits that one of ordinary skill in the art would be well-equipped to determine whether or not a claimed nucleic acid hybridizes to the complement of SEQ ID NO: 2 of the complement of a nucleotide sequence encoding SEQ ID NO: 7.

Applicant's arguments have been fully considered but are not found to be persuasive. The broad brush discussion of making and screening for PAP7 variants does not constitute a disclosure of a representative number of members. No such variants were made or shown to have activity. Only the full length nucleic acid sequence of SEQ ID NO: 2 and the amino acid

sequence of SEQ ID NO: 7 are disclosed. The specification's general discussion of making and screening for variants constitutes an invitation to experiment by trial and error. This disclosure is not adequate guidance, but is merely an invitation to the artisan to use the current invention as a starting point for further experimentation. Additionally, as was found in Ex parte Hitzeman, 9 USPQ2d 1821 (BPAI 1987), a single embodiment may provide broad enablement in cases involving predictable factors such as mechanical or electrical elements, but more will be required in cases that involve unpredictable factors such as most chemical reactions and physiological activity. See also In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970); Amgen Inc. v. Chugai Pharmaceutical Co. Ltd., 927 F.2d 1200, 1212, 18 USPQ2d 1016, 1026 (Fed. Cir.), cert. denied, 502 U.S. 856 (1991).

Although Applicant cites Sher et al. (J Biol Chem 274(49):35016-35022, 1999) and Kopchick et al. (U.S. Patent 5,350,836) as example references teaching how PAPs may be identified and how variants may be made, these references were cited by the Examiner to indicate the state of the art (i.e., growth factor polypeptide mutations alter the normal activity of the polypeptide). Sher et al. and Kopchick et al. made specific and limited mutations in the subject polypeptides that were examined. However, the instant claims encompass an infinite number of nucleic acid derivatives. Although the art (such as Sher et al. and Kopchick et al.) discloses the general methodology as to how to create variant polypeptides, undue experimentation would be required of the skilled artisan to generate the infinite number of PAP7 derivatives recited in the claims and to screen such for an activity. Such experimentation is considered undue.

The problem of predicting protein and DNA structure from sequence data and in turn utilizing predicted structural determinations to ascertain functional aspects of the protein and DNA is extremely complex. Certain positions in the amino acid sequence are critical to the protein's structure/function relationship, e.g. such as various sites or regions directly involved in binding, activity, and in providing the correct three-dimensional spatial orientation of binding and active sites. These regions can tolerate only relatively conservative substitutions or no substitutions.

Again, Sher et al. and Kopchick et al. report examples of growth factor polypeptide mutations which alter the normal activity of the polypeptide. Sher et al. disclose that keratinocyte growth factor (FGF-7) acts predominantly on cells of epithelial origin and regulates processes in embryonal and adult development, including cell growth, differentiation, cell migration, and repair of epithelial tissues (pg 35016, ¶ 1). Sher et al. demonstrate that point mutations in a loop of FGF-7 do not alter receptor binding affinity, but cause reduced mitogenic potency and reduced ability to induce receptor-mediated phosphorylation events (pg 35020-35021). Kopchick et al. disclose several antagonists of vertebrate growth hormone that differ from naturally occurring growth hormone by a single amino acid (column 2, lines 37-48). Therefore, based on the discussions above concerning the specific examples of structurally similar proteins that have different functions, the specification fails to teach the skilled artisan how to make and use biologically active PAP7 variants without resorting to undue experimentation to determine what the specific biological activities of the variants are.

Additionally, a single disclosed sequence does not support claims to any nucleic acid hybridizing to same, given the lack of guidance regarding what sequences would hybridize specifically to SEQ ID NO: 2, and not other, related sequences.

(ii) Applicant contends that Example 5 of the specification discloses how to determine the effect of a molecule on steroid biosynthesis. Applicant states that the specification discloses that fragments of PAP7 reduce the level of progesterone formation stimulated by saturating concentrations of hCG compared to control cells (pg 51, lines 10-14). Applicant argues that the PAP7 fragment is a competitor of native PAP7 in MA-10 cells, resulting in reduced cholesterol delivery to the IMM, thus regulating progesterone biosynthesis (pg 51, lines 18-21). Applicant also submits that in the prior art, the art worker was aware of assays to determine the ability of a claimed polypeptide to regulate progesterone biosynthesis and cholesterol delivery/transport. Applicant cites Papadopoulas et al. (1990) and Krueger and Papadopoulos (1990).

Applicant's arguments have been fully considered but are not found to be persuasive. Specifically, the specification's general discussion of making and screening for variants constitutes an invitation to experiment by trial and error. This disclosure is not adequate guidance, but is merely an invitation to the artisan to use the current invention as a starting point for further experimentation. Although the art discloses the general methodology as to how to create variant polypeptides, undue experimentation would be required of the skilled artisan to generate the infinite number of derivatives recited in the claims and possibly screen same for activity. As discussed in the previous Office Action and mentioned above, the specification discloses that *partial PAP7* transfectants significantly *reduce* the level of progesterone

production in MA-10 cells as compared with pSVzeo vector transfectants at a dose and time dependent manner (pg 47, lines 23-26; Fig 6). However, Li et al. (Molec Endocrinol 15(12): 2211-2228, 2001; a post-filing date reference by the instant inventors) teach that MA-10 cells transfected with *full-length PAP7* show an *increased* ability to form progesterone in response to hCG (pg 2219, col 1, 2nd full ¶; Figure 9A). Similarly, Li et al. disclose that transfection of MA-10 cells with full-length PAP7 followed by treatment with hCG results in higher production of pregnenolone, reflecting increased accumulation of cholesterol at the inner mitochondrial membranes (pg 2219, col 2, 1st full ¶). Li et al. also teach that transfection of MA-10 cells with the partial PAP7 sequence inhibit pregnenolone formation by 70% (pg 2219, 1st full ¶; Fig 10). Therefore, the full-length PAP7 protein and the partial PAP7 protein have opposite functions regarding progesterone production and pregnenolone formation (cholesterol accumulation). Undue experimentation would be required by one skilled in the art to generate an isolated nucleic acid sequence that is at least 90% identical to the nucleic acid sequence of SEQ ID NO: 2 and encodes a polypeptide that is capable of regulating progesterone biosynthesis, impairs cholesterol delivery, or increases cholesterol delivery. The skilled artisan must also resort to trial and error experimentation to screen all possible derivatives for such activities. Such trial and error is considered undue. Although amino acids 228-445 of SEQ ID NO: 7 are required for a decrease in progesterone production as evidenced by Li et al., undue experimentation would be required of the skilled artisan to determine which amino acids impart the function of full length PAP7 (e.g., increased progesterone biosynthesis). There is little or no guidance in the specification indicating which amino acids are required for the biological activity of full-length PAP7 or a protein encoded by an isolated nucleic acid sequence that is 90% identical to SEQ ID NO: 2.

There is also no guidance in the instant specification as to the specific amino acid sequence that comprises the partial PAP7 protein. Additionally, a large quantity of experimentation would be required by one skilled in the art to generate and screen for nucleic acid molecules that encode a polypeptide that impairs cholesterol delivery, increases cholesterol delivery, or regulates progesterone biosynthesis and hybridizes to the complement of the nucleic acid of SEQ ID NO: 2.

(iii) Applicant asserts that the specification clearly discloses that PAP7 mediates cholesterol delivery (pg 34, lines 15-17). Applicant indicates that there is no requirement for a working example to fulfill the requirement of § 112(1). Applicant argues that if the invention is disclosed so that one of ordinary skill in the art can practice the claimed invention, even if the practice would include routine screening or some experimentation, Applicant has complied with the requirements of § 112(1). Applicant cites *In re Robins*, 166 USPQ 552 (CCPA 1970), *In re Angstadt*, 190 USPQ 214(C.C.P.A. 1976) and *Ex parte Jackson*, 217 USPQ 804(Bd. Appl 1982).

Applicant's arguments have been fully considered but are not found to be persuasive. Specifically, the specification teaches at pg 34, lines 15-17 that "the PAPS identified in this application were discovered due to their ability to associate with PBR, and may play a role in the proper targeting, function, expression, or stability of PBR". It is not clear to the Examiner how this general disclosure specifically teaches that PAP7 mediates cholesterol delivery. The specification also teaches at pg 3, lines 10-23 that PBR ligands stimulate pregnenolone formation by increasing the rate of cholesterol transfer from the outer to the inner mitochondrial membrane.

However, there is no guidance in the specification teaching that PAP7 is a ligand or that it mediates, inhibits, or stimulates cholesterol transport.

Additionally, although Applicant need to not actually have reduced the invention to practice prior to filing the application, the lack of a working example is only one factor to be considered, especially in a case involving an unpredictable art (MPEP § 2164.02). The specification teaches little or no guidance indicating that PAP7 regulates PBR activity in cholesterol transport or how to test for such PAP7 activity. This is merely an invitation for the artisan to use the current invention as a starting point for further experimentation. Such experimentation is considered undue. A specification may be enabling even though some experimentation is necessary, but the amount of experimentation, however, must not be unduly extensive. According to MPEP § 2164.06, "the guidance and ease in carrying out an assay to achieve the claimed objectives may be an issue to be considered in determining the quantity of experimentation needed". Additionally, the post-filing date reference of Li et al. (cited above) teaches an assay to determine the role of PAP7 in cholesterol transport. Li et al. disclose that transfection of MA-10 cells with full-length PAP7 followed by treatment with hCG results in higher production of pregnenolone, reflecting increased accumulation of cholesterol at the inner mitochondrial membranes (pg 2219, col 2, 1st full ¶). Li et al. also teach that transfection of MA-10 cells with the partial PAP7 sequence inhibit pregnenolone formation by 70% (pg 2219, 1st full ¶; Fig 10). Therefore, the full-length PAP7 protein and the partial PAP7 protein have opposite functions regarding pregnenolone formation (cholesterol accumulation) and one skilled in the art would not be able to predict which of the numerous claimed variants increase cholesterol delivery or impair cholesterol delivery.

Proper analysis of the Wands factors was provided in the previous Office Action. Due to the large quantity of experimentation necessary to generate the infinite number of derivatives recited in the claims and possibly screen same for activity, the lack of direction/guidance presented in the specification regarding which structural features are required in order to provide activity, the absence of working examples directed to same, the complex nature of the invention, the state of the prior art which establishes the unpredictability of the effects of mutation on protein structure and function, and the breadth of the claims which recite broad structural and functional limitations, undue experimentation would be required of the skilled artisan to make and/or use the claimed invention in its full scope.

3. Claims 43-44, 46-47, 57-64, 69-82 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. The basis for this rejection is set forth at pg 8-11 of the previous Office Action of 23 June 2004, at pg 9-11 of the Office Action of 25 September 2003, and at pg 9-12 of the Office Action of 10 April 2003.

The claims are directed to an isolated nucleic acid sequence that is at least 90% identical to a nucleotide sequence encoding SEQ ID NO: 7, which isolated nucleic acid encodes a polypeptide that is capable of regulating progesterone synthesis, or the complement thereof. The claims recited an isolated nucleic acid sequence that is at least 90% identical to a nucleotide sequence encoding SEQ ID NO: 7, which isolated nucleic acid encodes a polypeptide that impairs cholesterol delivery. The claims recite a process of producing a peripheral-type

benzodiazepine-associated protein. The claims recite a vector comprising a heterologous promoter linked the PAP7 nucleotide sequence and reagents comprising the nucleic acid. The claims recite an isolated nucleic acid that hybridizes to the complement of SEQ ID NO: 2 or to the complement of the nucleotide sequence that encodes the polypeptide of SEQ ID NO: 7. The claims recited an isolated nucleic acid sequence that is at least 90% identical to a nucleotide sequence encoding SEQ ID NO: 7, which isolated nucleic acid encodes a polypeptide that increases cholesterol delivery.

Applicant's arguments (27 September 2004), as they pertain to the rejections have been fully considered but are not deemed to be persuasive for the following reasons.

(i) Applicant asserts that the instant specification discloses isolated nucleic acid having PAP sequences including those encoding SEQ ID NO: 7 or having SEQ ID NO: 2. Applicant argues that the specification discloses that the invention includes nucleic acids that hybridize to the complement of those sequences under stringent hybridization conditions., and nucleic acids that are 90% or more identical thereto. Applicant contends that the claimed nucleic acids have particular structural and functional features. Applicant concludes that the claims recite functional characteristics coupled with a known or disclosed correlation between function and structure and so are in compliance with the written description requirement under 35 U.S.C. § 112, first paragraph.

Applicant's arguments have been fully considered but are not found to be persuasive. Specifically, Applicant has not provided evidence to demonstrate that the skilled artisan would be able to envision the detailed structure of the infinite number of polynucleotides recited in the claims. To provide adequate written description and evidence of possession of a claimed genus,

the specification must provide sufficient distinguishing identifying characteristics of the genus. The factors to be considered include disclosure of compete or partial structure, physical and/or chemical properties, functional characteristics, structure/function correlation, methods of making the claimed product, or any combination thereof. In this case, the only factor present in the claims is a partial structure in the form of a recitation of percent identity. There is not even identification of any particular portion of the structure that must be conserved. Accordingly, in the absence of sufficient recitation of distinguishing identifying characteristics, the specification does not provide adequate written description of the claimed genus. The description of one full length PAP7 polynucleotide and polypeptide in the specification of the instant application is not a representative number of embodiments to support the description of an entire genus of functionally equivalent polynucleotides and polypeptides which incorporate all mutants, derivatives, and variants with at least 90% sequence identity to the polypeptide of SEQ ID NO: 7 or the nucleic acid of SEQ ID NO: 2 that impair cholesterol delivery, increase cholesterol delivery, or regulate progesterone biosynthesis. Furthermore, the broad brush discussion of making or screening for variants does not constitute a disclosure of a representative number of members. No such variants were made or shown to have activity. The specification's general discussion of making and screening for variants constitutes an invitation to experiment by trial and error. Such does not constitute an adequate written description for the claimed variants.

Additionally, with regard to claims 46, 47, and 79, simply reciting hybridization conditions in the claims does not yield adequate written description of the polynucleotides encompassed. The claims encompass an infinite number of polynucleotides that hybridize to the nucleic acid sequence of SEQ ID NO: 2 or to the nucleic acid sequence that encodes the

polypeptide of SEQ ID NO: 7. These polynucleotides may be structurally and functionally divergent from the polynucleotide of SEQ ID NO: 2 and from the polynucleotide encoding the polypeptide of SEQ ID NO: 7.

4. Claims 44, 47, 61-64, and 73-79, and 81-82 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a new matter rejection. The basis for this rejection is set forth at pg 11-12 of the previous Office Action (23 June 2004).

Claim 44 is directed to an isolated nucleic acid comprising a nucleic acid sequence that is at least 90% identical to the sequence of the nucleic acid sequence of claim 41 and encodes a polypeptide that impairs cholesterol delivery. Claim 47 recites an isolated nucleic acid that encodes a polypeptide that impairs cholesterol delivery and hybridizes to the complement of the nucleic acid of claim 41(a) or 41(b). The claims also recite vectors comprising the nucleic acids, host cells comprising the vectors, a process of producing a PAP, and diagnostic agents comprising the nucleic acids. Claim 77 recites an isolated nucleic acid comprising a nucleic acid sequence that is at least 90% identical to a nucleotide sequence encoding SEQ ID NO: 7, which isolate nucleic acid encodes a polypeptide that increases cholesterol delivery, or the complement thereof. Claim 78 recites an isolate nucleic acid sequence that is at least 90% identical to SEQ ID NO: 2 and encodes a polypeptide that increases cholesterol delivery. Claim 82 recites an isolate nucleic acid comprising a nucleic acid sequence encoding SEQ ID NO: 7 and variants thereof that are at least 90% identical, which isolated nucleic acid encodes a polypeptide that

facilitates cholesterol transport from the outer mitochondrial membrane to the inner mitochondrial membrane.

Applicant's arguments (27 September 2004), as they pertain to the rejections have been fully considered but are not deemed to be persuasive for the following reasons.

Applicant asserts that the application discloses that PAP7 interacts with PBR (pg 5, lines 19-24; pg 34, lines 14-17) and regulates PBR activity in cholesterol transport (pg 49, lines 19-22). Applicant also indicates that it is disclosed that the function or stability of PBR and associated PAP7 can be manipulated to increase cholesterol transport into cells (pg 35, line 32 through pg 36, line 10; pg 50, lines 11-17). Applicant contends that PAP7 can be used to competitively bind PBR and block normal PBR function, i.e. impair cholesterol transport into the IMM (pg 51, lines 18-26).

Applicant's arguments have been fully considered but are not found to be persuasive. PAP7 is not disclosed by the instant specification to be an agent or a drug and the specification clearly discloses that "*an agent or drug* which results in an increase in function or stability of PBR *and its associated PAPs* can be used to increase cholesterol transport into cells" (pg 35, line 32 through pg 36). The Examiner interprets this sentence in the specification to indicate that the agent/drug increases PBR function which in turn, increases cholesterol transport. There is no indication in this particular sentence or the rest of the specification that PAP7 itself increases or impairs cholesterol delivery. The specification also teaches that the PAP7 *fragment* competitively prevents PBR from interacting with the endogenous PAP7 and thereby blocks the normal function of PBR" (pg 51, lines 18-26). However, the specification of the instant application does not directly associate the activity of "impairing cholesterol delivery/transport"

with the PAP7 polypeptide or its variants. Similarly, the specification as originally filed does not provide adequate written description for an isolated nucleic acid or variants that encode a PAP7 polypeptide that increases cholesterol delivery/transport. The specification as originally filed also does not provide adequate written description for an isolated nucleic acid or variants that encode a PAP7 polypeptide that facilitates cholesterol transport from the outer to the inner mitochondrial membrane. It is not expressly asserted, nor does it flow naturally from the specification. It is noted that at pg 7 of the response of 27 September 2004, Applicant indicated where support in the specification for the newly added claims could be located. However, as mentioned previously, the disclosure does not specifically associate the PAP7 polypeptide and increasing cholesterol delivery or facilitating cholesterol transport from the outer to the inner mitochondrial membrane.

Allowable Subject Matter

Claims 10-16, 41, and 48-52 are allowable.

Conclusion

Claims 43-44, 46-47, 57-64, and 69-82 are rejected.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Bridget E. Bunner whose telephone number is (571) 272-0881. The examiner can normally be reached on 8:30-4:30 M-F.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Brenda Brumback can be reached on (571) 272-0961. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Elizabeth C. Kemmerer

BEB
Art Unit 1647
07 December 2004

ELIZABETH KEMMERER
PRIMARY EXAMINER